and height. Our purpose was to evaluate the spirometers, not the subjects.

Dr. Sharp describes the potential problem of testing spirometric devices in series and thereby creating a back or loading pressure. This was initially examined in our study by testing each device alone and then together in series using a calibrated syringe volume of air delivered at various flows. No significant difference could be measured. Similarly, ten subjects were first tested using the three devices alone and in series; and, again, no important difference in flows resulted. The one-way valves used to obtain unidirectional flow eliminated any opportunity for back pressure.

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Systolic Anterior Motion of the Mitral Valve Due to Hypovolemia and Anemia

To the Editor:

In the May 1976 issue of Chest, Bulkley and Fortuin reported a case of echocardiographic systolic anterior motion of the mitral valve suggesting left ventricular outflow obstruction in the absence of hypertrophic cardiomyopathy. I recently had a similar experience that I would like to report here.

A 74-year-old white woman was transferred to the University of California-Los Angeles Medical Center because of diarrhea, vomiting, and hypotension. The patient was in excellent health until three months prior to admission, when she began to experience frequent episodes of diarrhea. Two weeks prior to admission, she was admitted to another hospital because of worsening of the diarrhea, nausea, and vomiting. The patient was dehydrated and hypotensive, and a loud systolic murmur was noted for the first time. Over the next two weeks, she developed edema of the lower extremities, hepatosplenomegaly, and jaundice. On the day prior to the transfer, the patient became confused and oliguric.

On admission the blood pressure was 70/50 mm Hg, and the pulse rate was 120 beats per minute. The patient was slightly confused and jaundiced. The neck veins were flat. The chest was clear to percussion and auscultation. The point of maximum cardiac impulse was not displaced, and the heart sounds were normal. There was a grade-3/6 long systolic murmur loudest at the apex, with radiation to the base and axilla; no gallops were heard. The abdomen was distended, the liver and spleen were enlarged and firm, and there was marked edema of the abdominal wall, flanks, and lower extremities.

The hematocrit reading was 23 percent. Concentrations of bilirubin and creatinine were elevated. The electrocardiogram was normal. A chest x-ray film showed an infiltrate in the left lower lobe, with a normal cardiac size. An echocardiogram (Fig 1) showed normal thickness of the septum and the posterior left ventricular wall, with a small chamber size. There was a systolic anterior motion of the mitral valve, contacting the septum as typically seen in idiopathic hypertrophic subaortic stenosis.

Therapy with fluid replacement, blood transfusions, antibiotics, steroids, and hemodialysis was instituted, with initial improvement. There was softening of the murmur with fluid
reduction. After the first day of hospitalization, the patient deteriorated very rapidly, with progressive hepatomegaly, jaundice, and infradiaphragmatic edema. There was also complicating thrombocytopenia with bleeding. The patient died on the seventh day of hospitalization.

On postmortem examination, there was hepatocellular carcinoma with multiple intra-abdominal metastases and thrombosis of the inferior vena cava. The heart weighed 275 gm and was normal, with normal thickness of the septum and left ventricular free wall. Microscopic study showed normal orientation of the myofibers and no evidence of hypertrophy.

**DISCUSSION**

A systolic anterior motion of the mitral valve has been known to occur in hypertrophic cardiomyopathy, and it is evidence for outflow obstruction; its mechanism is still not well understood. We have also observed that ectopic chordae tendineae can also produce an abnormal systolic anterior motion of the mitral valve with functional outflow obstruction in the absence of hypertrophic cardiomyopathy (unpublished data). Bulkley and Fortuin reported a case of typical echocardiographic mitral systolic anterior motion in a patient with a normal heart. They postulated that hypovolemia and catecholamine stimulation were responsible for such an abnormality.

Several investigators have shown that hemorrhagic shock and infusion of catecholamines may produce functional intraventricular obstruction. In our case the combination of hypovolemia and anemia seem to have caused the echocardiographic abnormality. Although in our case, as in the one reported by Bulkley and Fortuin, no pressure recordings were made, the association of a loud systolic murmur and the systolic anterior motion in the echocardiogram suggests an outflow gradient. The absence of left ventricular hypertrophy, the finding of a perfectly normal mitral leaflet with no thickening, and the history of a recent murmur all suggest that the functional obstruction was indeed related to the circumstances of terminal illness, namely, hypovolemia and anemia. I would like to subscribe to the speculation of Bulkley and Fortuin that a small, underfilled left ventricle with increased contractility due to infusion of catecholamines or, as in our case, to anemia may produce a systolic murmur and abnormal anterior systolic motion of the mitral valve. The physiologic change in the left ventricle simulates hypertrophic cardiomyopathy with obstruction and suggests that the approximation of the mitral valve to the interventricular septum, with increased contractility and high velocity of the ejection flow, are responsible for the functional obstruction, as has been suggested previously.

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**REFERENCES**


**To the Editor:**

Levisman has described another case of systolic anterior motion of the mitral valve unrelated to hypertrophic cardiomyopathy. We have also seen five additional patients with echocardiographic systolic anterior motion of the mitral valve and typical auscultatory and peripheral pulse abnormalities characteristic of idiopathic hypertrophic subaortic stenosis in the absence of hypertrophic cardiomyopathy as determined by myocardial imaging, cardiac catheterization, or autopsy or some combination of the three. The common denominator in these patients, including the additional case reported by Levisman, was a relatively contracted left ventricular cavity and an increased myocardial contractility, both of which may be transient phenomena. These cases are especially important, as they indicate the limitations of the echocardiographic criteria for diagnosis of hypertrophic cardiomyopathy, and they suggest that one must be careful not to make the erroneous diagnosis of a chronic myocardiopathy with autosomal dominant inheritance and a potentially grave prognosis in any asymptomatic individual on the basis of an echocardiogram alone.

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**REFERENCE**


**Coronary Arterial Aneurysm vs Poststenotic Dilatation**

**To the Editor:**


Nevertheless, I was a bit disturbed by the classification of some of the abnormalities illustrated as aneurysms. In spite of the definition of aneurysms as...